SLR:slr:dm 12/4/02 154945
PATENT

Attorney Reference Number 4239-62631
Application Number 09/634,369

Claim 33: Page 2, line 28 – page 3 line 1; page 4, lines 7-11;

Claim 34: Original claim 15;

Claims 35 and 40: Page 3, lines 4-5; page 9, lines 1-3; page 35, line 10 – page 36, line 24; and

Claims 36 - 39: Original claims 16 –19, respectively.

Original claims 15, 16, 17, 18, and 19, which depended (directly or indirectly) from claim 10, have been re-written as new claims 34, and 36-39, respectively, to depend from allowed claim 28. The claims were re-written as new claims, instead of amended to depend from claim 28, for the Examiner's convenience. Therefore, no new matter will be added, and no new issues will be raised, by these claims and Applicant requests that they be entered.

New claims 33, 35 and 40, which depend from allowed claim 28, are clearly supported in the specification as noted above. Since these claims are supported by the specification, and depend from an allowed claim, no new matter will be added, and no new issues will be raised, by these claims and Applicant requests that they be entered.

In conclusion, this amendment places the application in condition for immediate allowance, and Applicants therefore respectfully request that it be entered. If the Examiner believes any minor matters remain to be resolved, she is encouraged to contact the undersigned.

Respectfully submitted,

KLARQUIST SPARKMAN, LLP

By _____
Sheree Lynn Rybak, Ph.D.
Registration No. 47,913

One World Trade Center, Suite 1600
121 S.W. Salmon Street
Portland, Oregon 97204
Telephone: (503) 226-7391
Facsimile: (503) 228-9446

SLR:slr:dm 12/4/02 154945.doc
PATENT

Attorney Reference Number 4239-62631
Application Number 09/634,369

Marked-up Version of Amended Claims
Pursuant to 37 C.F.R. §§ 1.121(b)-(c)

10. (Cancel) [A method for treating inflammation or an immunological disorder, comprising:

administering to a subject a therapeutically effective amount of a composition of matter selected from the group consisting of epoxyeicosatrienoic acids (EETs), epoxyeicosatrienoic acid metabolic products, epoxyeicosatrienoic acid and dihydroxyeicosatrienoic acid analogs, and combinations thereof,

wherein the therapeutically effective amount of the composition of matter reduces inflammation or the immunological disorder in the subject.]

11. (Cancel) [The method of claim 10, wherein the inflammation is caused by cardiovascular disease.]

12. (Cancel) [The method of claim 10, wherein the inflammation is caused by a rheumatologic disorder.]

13. (Cancel) [The method of claim 10, wherein the inflammation is caused by atherosclerosis.]

14. (Cancel) [The method of claim 10, wherein the inflammation is caused by an autoimmune disorder.]

15. (Cancel) [The method of claim 10, wherein the wherein the administration comprises producing EETs from a cytochrome P450 epoxygenase.]

16. (Cancel) [The method of claim 15, wherein the cytochrome P450 epoxygenase is selected from the group consisting of the CYP1A, CYP2B, CYP2C, CYP2E, and CYP2J enzymes.]

17. (Cancel) [The method of claim 16, wherein the CYP2J enzyme is a mammalian homologue of CYP2J2.]

18. (Cancel) [The method of claim 16, wherein the homologue is human CYP2J2.]

19. (Cancel) [The method of claim 16, wherein the homologue is rat CYP2J3 or mouse CYP2J5.]

21. (Cancel) [The method of claim 10, further comprising:
administering an epoxide hydrolase inhibitor to the subject.]

25. (Cancel) [A method for preventing inflammation or an immunological disorder, comprising:
administering to a subject a prophylactically effective amount of a composition of matter selected from the group consisting of epoxyeicosatrienoic acids (EETs), epoxyeicosatrienoic acid metabolic products, epoxyeicosatrienoic acid and dihydroxyeicosatrienoic acid analogs, and combinations thereof,
wherein the prophylactically effective amount of the composition of matter prevents inflammation or the immunological disorder in the subject.]

26. (Cancel) [A method for inhibiting expression of cell adhesion molecule in an endothelial cell, comprising:
contacting an endothelial cell with an effective amount of a composition of matter selected from the group consisting of epoxyeicosatrienoic acids (EETs), epoxyeicosatrienoic acid metabolic products, epoxyeicosatrienoic acid and dihydroxyeicosatrienoic acid analogs, and combinations thereof,
wherein the effective amount of the composition of matter is sufficient to inhibit expression of cell adhesion molecule VCAM-1 by the endothelial cell.]

27. (Cancel) [A method for modulating NF-κB activity in a cell, comprising:
contacting a cell with an effective amount of a composition of matter selected from the group consisting of epoxyeicosatrienoic acids (EETs), epoxyeicosatrienoic acid metabolic products, epoxyeicosatrienoic acid and dihydroxyeicosatrienoic acid analogs, and combinations thereof,
wherein the effective amount of the composition of matter is sufficient to inhibit IκB kinase (IKK) in the cell.]

33. (New) The method of claim 28, wherein contacting a cell comprises administration of EETs, epoxyeicosatrienoic acid metabolic products, epoxyeicosatrienoic acid, dihydroxy icosatrienoic acid

analogs, and combinations thereof to a subject.

34. (New) The method of claim 33, wherein the wherein the administration comprises producing EETs from a cytochrome P450 epoxygenase.

35. (New) The method of claim 34, wherein the EET is [11,12]-EET, [14,15]-EET, or combinations thereof, and wherein the epoxyeicosatrienoic acid metabolic product is [11,12]-DHET.

36. (New) The method of claim 34, wherein the cytochrome P450 epoxygenase is selected from the group consisting of CYP1A, CYP2B, CYP2C, CYP2E, and CYP2J enzymes.

37. (New) The method of claim 36, wherein the CYP2J enzyme is a mammalian homologue of CYP2J2.

38. (New) The method of claim 37, wherein the mammalian homologue is human CYP2J2.

39. (New) The method of claim 37, wherein the mammalian homologue is rat CYP2J3 or mouse CYP2J5.

40. (New) The method of claim 28 wherein the EET is [11,12]-EET or [14,15]-EET, and wherein the epoxyeicosatrienoic acid metabolic products is [11,12]-DHET.